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### *Reply to the Editor:*

We appreciate the response by Stöllberger, Schneider, and Finsterer to our work[1] and thank them for their valuable comments.

As they mentioned, this series in dogs was too small to confirm the long-term safety and tissue response to the atrial exclusion device (AED). However, the main purpose of this series was to assess the feasibility of the device implant during a beating heart procedure, and thus we did not include a control group or an atrial fibrillation (AF) animal model, which would have been helpful to evaluate the safety and efficacy of this device.

Because of the main goal of this study series, we also did not analyze biochemistry levels such as serum natriuretic peptide, troponin, and creatine kinase, nor did we obtain hemodynamic data derived from cardiac catheterization with the exception of left atrial (LA) pressure, heart rate, and systemic arterial pressure. However, we performed echocardiographic evaluations at baseline, after AED implant, and at follow-up. The pulmonary venous flow, transmitral flow, and tissue Doppler imaging of the mitral annular motion, which provide additional information regarding atrial and ventricular filling and function,[2,3] were evaluated to assess the influence of left atrial appendage (LAA) exclusion on LA function. The Doppler pulmonary venous flow data suggested that LAA exclusion may affect LA reservoir function without affecting left ventricular diastolic function. After we obtain echocardiographic data from our next series of animals, we intend to report more detailed echocardiographic evaluation results in the near future.

With respect to the effect of device implant on flow through the left circumflex artery, although specific evaluations including coronary angiography and left circumflex flow measurement with a flow probe were not performed in this series, no macroscopic findings of myocardial infarction at explant surgery or gross examination were found.

As also mentioned, LAA exclusion may cause adverse effects due to lack of serum natriuretic peptide. In this series, there were no clinical signs of heart failure, including general fatigue or significant changes in the weights and heart rates of any dog, throughout the study.

To date, there have been no reports showing clear evidence of a role of LAA exclusion on stroke prevention for patients with nonvalvular AF. However, considering that LA thrombi in more than 90% of cases of nonvalvular AF are located in the LAA[4] and surgical LAA ligation or excision does not appear to have clinically important deleterious effects according to extensive study on the Maze procedure by Cox and associates,[5] LAA exclusion should be considered one of the most important therapeutic options, especially for patients with AF who are not eligible for any anticoagulation therapy.

We agree with the potential concerns related to hemodynamic and neurohumoral consequences after LAA exclusion. In preparation for a clinical AED application, further evaluations using both a control and AF animal model will definitely be required to address the following points: (1) competency of LAA exclusion with various LAA sizes, (2) long-term stability of the AED, (3) potential LA thrombus formation due to the device implant in an AF model, and (4) neurohumoral effects of LAA exclusion. We hope these further evaluations will provide us with clearer information that elucidates the exact role of LAA exclusion.

We continue to work on this subject and look forward to publishing more information on the AED, the implantation procedure for LAA exclusion, and its effects on physiologic function.

*Keiji Kamohara, MD*[a]
*Kiyotaka Fukamachi, MD, PhD*[a]
*A. Marc Gillinov, MD*[b]
*Department of Biomedical Engineering*[a]
*Department of Thoracic and Cardiovascular Surgery*[b]
*Cleveland Clinic*
*Cleveland, OH 44195*

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## Is it really the number of clamps that is responsible for worse postbypass neurological outcomes?

### *To the Editor:*

I read with interest the article by Dr Hammon and colleagues[1] comparing varying levels of aortic manipulation in bypass surgery with regard to postoperative neurobehavioral outcomes. Even as the results are in some concurrence with certain previous trials in this direction, I do believe that this report requires careful review before its recommendations can be accepted.

First, the multiple aortic clamping group had greater age, incidence of hyperlipidemia, and aortic fibrillation than the single aortic clamping group, 3 independent predictors of postoperative stroke.[2] Second, the authors were unable to demonstrate any significantly worse result in the 2 groups, apart from the neuropsychologic deficit at 6-month follow-up. Recent prospective neuropsychologic testing indicates that these late neurocognitive deficits are likely to be caused by established risk factors for cerebral vasculopathy not having anything to do with the bypass procedure itself.[3]

It is therefore arguable that the mere reduction in the number of clamps would have a significant effect on patient neurological profile after bypass surgery, considering that there exists already conflicting evidence, with no additional benefit of reduction in clamp number being recorded previously.[4] Consensus in the literature has